EXHIBIT E

1: J Infect Dis. 1989 Feb;159(2):239-47.

Links

Interleukin-2 protects neonatal mice from lethal herpes simplex virus infection: a macrophage-mediated, gamma interferon-induced mechanism.

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Administration of human recombinant interleukin-2 (IL-2) protected neonatal mice from a lethal herpes simplex virus (HSV) infection. Protection was not associated with viral antibody production, enhanced natural killer cell cytotoxicity, or intrinsic resistance of macrophages to viral infection. Protection was associated with increased macrophage-mediated antiviral antibody-dependent cellular cytotoxicity (ADCC). Spleen cells from IL-2-treated neonatal mice and from neonatal mice that were treated in vitro with IL-2 transferred protection to neonatal mice. These cells, by adherence, silica, and asialo GM 1 antibody treatment, were shown to be macrophages. IL-2 treatment in vitro enhanced the neonatal macrophages' ADCC function and superoxide release. Similar protection was induced by gamma interferon (IFN-gamma)-treated spleen cells. Antibody to IFN-gamma ablated both IFN-gamma- and IL-2-induced protection by adherent spleen cells. Thus, IL-2-mediated protection against murine neonatal HSV infection was affected by stimulated macrophage activity, via helper T cell-produced IFN-gamma.

PMID: 2492588 [PubMed - indexed for MEDLINE]

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